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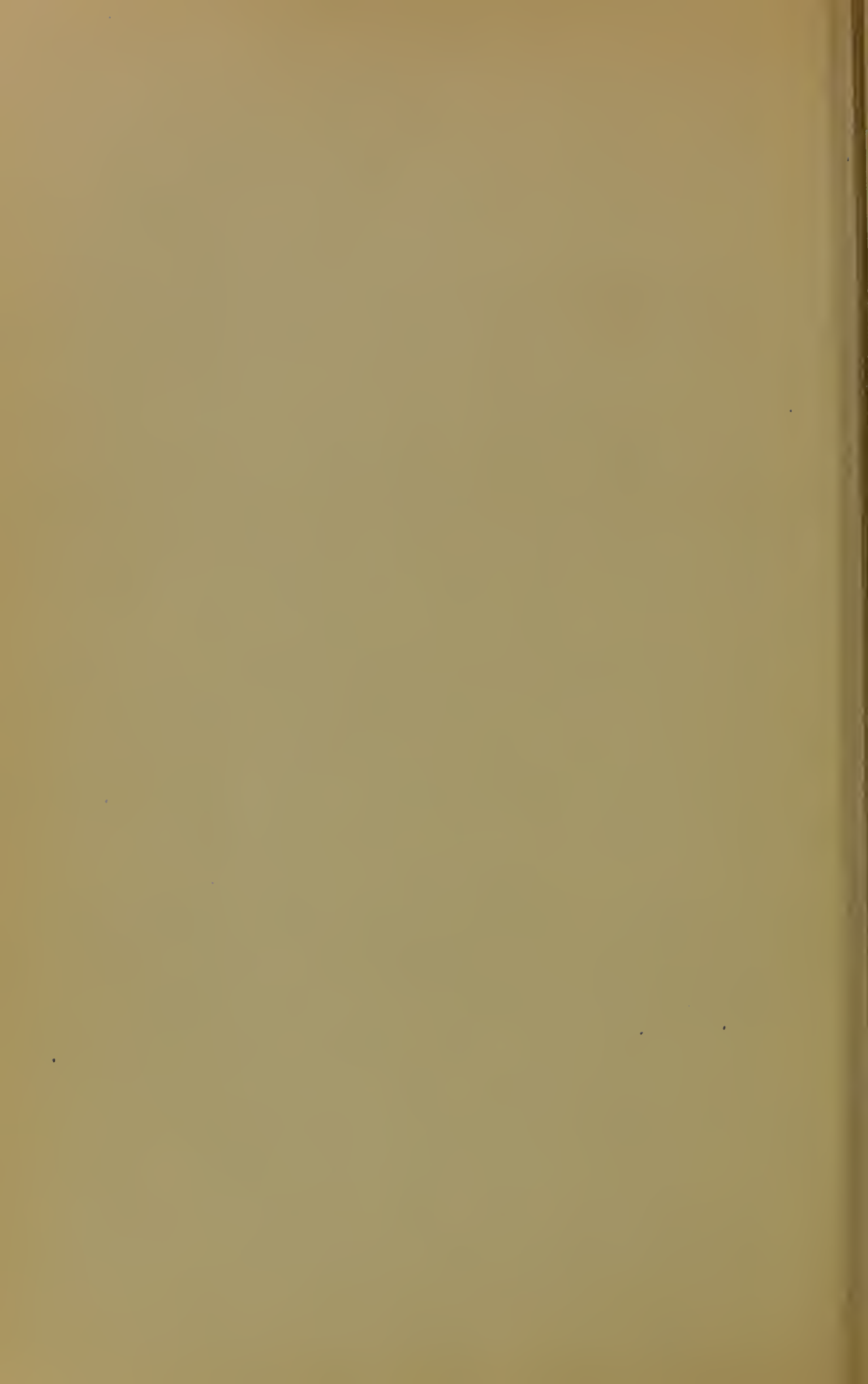
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FATAL HÆMOPTYSIS IN A CHILD AGED 4 YEARS.

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FATAL HÆMOPTYSIS IN A CHILD AGED 4 YEARS.*

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A BOY, aged 4 years, was admitted to the Grove Fever Hospital on February the 19th, 1914, certified to be suffering from whooping cough. Measles, in December, 1912, was stated to be the only previous illness, but since November, 1913, he had appeared generally unwell and subject to attacks of abdominal pain and vomiting.

Condition on admission: The child was in a very ill-nourished and wasted state. The cough was paroxysmal, but at no time during his stay in hospital was he heard to whoop. Rhonchi and râles could be heard all over both lungs, but no areas of dulness could be marked out. The abdomen was distended and the superficial abdominal veins were dilated. The spleen could be palpated below the level of the costal margin and the area of liver dulness was slightly increased downwards. No enlargement of the lymphatic glands was detected. The temperature rose nightly to between 102° and 103° , but fell to nearly normal in the morning. The pulse-rate was rarely below 120 and the respiration varied 30 and 50.

On March the 15th a specimen of sputum was with difficulty obtained from the mouth, but no tubercle bacilli were seen. Von Pirquet's reaction, performed on April the 3rd, was also negative, a fact which is of interest in view of the subsequent post-mortem findings. (A positive reaction, however, was found in the patient's sister, aged 2 years, who was admitted on the same day with the certificate of whooping cough, and died on May the 21st of tuberculous meningitis—a terminal episode of what was probably a generalised infection. An autopsy could not be obtained, but three

* A paper read at the Section for the Study of Disease in Children of the Royal Society of Medicine, on May the 22nd, 1914.

days before death some cloudy cerebro-spinal fluid was obtained on which Dr. Cartwright Wood, Bacteriologist to the Board, reported as follows: "We killed two guinea-pigs, inoculated with the cerebro-spinal fluid, and found both animals markedly affected with tuberculosis. We were able to demonstrate the presence of tubercle bacilli in the caseous material at the site of injection.")

A differential blood-count on April the 14th showed: Polymorphonuclears, 54 per cent., small mononuclears, 40 per cent., large mononuclears, 6 per cent. Very little change occurred in the child's general condition, but on April the 13th impairment of resonance was noted below the left ninth rib behind and extending round into the left axilla; over this area the breathing was bronchial in character. During the evening of April the 24th a very little blood-stained sputum was expectorated, and at 8.30 p.m. on the 25th, after a comfortable day during which the child seemed rather brighter than usual, a sudden copious hæmoptysis occurred, death ensuing almost immediately.

Necropsy, April the 26th.—On pressing on the trachea and œsophagus a quantity of dark blood was expelled from the mouth and nostrils. The tracheo-bronchial glands were much enlarged, obviously tuberculous and some of them caseating. The left pleura was thickened and firmly adherent to the chest-wall and diaphragm. The upper lobe of the left lung was studded with tuberculous deposits. The lower lobe was solid, small pieces of it sinking in water, and presented a cavity about the size of a walnut, completely filled with recent blood-clot. The whole of the right lung was a mass of tuberculous nodules. The liver was enlarged, fatty and studded with tubercles. The spleen was considerably enlarged and contained tuberculous deposits of a fair size. Tubercles were also seen in the kidneys, basal meninges and grey matter of the cerebral cortex. The heart was empty and normal in size and appearance. The mesenteric glands were enlarged and the stomach contained a large quantity of altered blood but showed no sign of any lesion in the mucous membrane.

This case is of interest in the first place as being an example of general tuberculosis with the primary focus in the tracheo-bronchial glands producing a paroxysmal cough, simulating whooping cough, and in the second place owing to the rarity of hæmoptysis in so young a child. Although, as in seven out of twenty-three cases collected by Meusnier which came to autopsy the actual bleeding vessel was not definitely identified, it is presumable that the hæmoptysis occurred through the rupture of a branch of the pulmonary artery into the

cavity found in the left lower lobe. Death was ascribed to asphyxiation rather than to the amount of blood lost.

A somewhat similar case occurred at the same hospital nearly three years previously. A boy, aged 4 years, was admitted with the certificate of whooping cough on June the 8th, 1911. His cough was sometimes paroxysmal, but no whoop occurred during his six months' stay in hospital. The signs and symptoms were those of chronic pulmonary tuberculosis, and the paroxysmal cough suggested affection of the tracheo-bronchial glands. Von Pirquet's reaction performed on July the 19th was positive. Two attacks of profuse hæmoptysis occurred on December the 20th, and death took place on the same day. No autopsy could be obtained.

Meusnier (3) in 1892 collected thirty-three cases of hæmoptysis in children aged from 8 days to 14 years, but only sixteen of these were in children under 5 years. Of these sixteen all but three were tuberculous and all but one—a case of influenzal bronchitis—were fatal. The other two non-tuberculous cases occurred in new-born infants, in one of whom the lungs seem to have been digested by the inspiration of gastric juice during vomiting, while in the other the hæmoptysis was due to pulmonary thrombosis of syphilitic origin.

Magruder (2) in 1908 described a case in a negro boy, aged 3 years, very similar to our own, and referred to four other cases aged from 7 months to 3½ years, in which fatal hæmoptysis was due to pulmonary tuberculosis. Other cases of fatal hæmoptysis in children due to pulmonary tuberculosis have since been reported by Brown (1) in a girl of 3 years and by Thomas (5) in an infant of 3 months. It would thus appear that hæmoptysis occurring in young children is in the majority of cases due to pulmonary tuberculosis. It is as a rule a sudden and fatal event. In only five of Meusnier's twenty-nine fatal cases had the terminal hæmoptysis been preceded by any spitting of blood. In this respect the slight premonitory hæmoptysis which occurred in our case was exceptional.

The hæmorrhage may be caused by the rupture of an aneurysm or of a branch of the pulmonary artery into a cavity within the lung, either in the apex or the base, or by the breaking down of tracheo-bronchial glands surrounding a vessel and causing its rupture into a neighbouring bronchus. The fact that the mouth, pharynx and lower air-passages are as a rule filled with blood, demonstrates the mode of death in these cases. The stomach is generally found to contain much blood, suggesting at first sight hæmetemesis, but no lesions can be detected in the mucous membrane. Other causes of fatal hæmoptysis in children besides tuberculosis may be gangrene

of the lung, ulceration of the neck-vessels in scarlet fever, or gangrenous angina or hæmorrhage in ulcerative sore throat, an example of which was reported by one of us to this section during the last session (4).

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- (5) THOMAS, E. H.—'Brit. Med. Jour.,' 1909, i, p. 1356.